International Journal of Clinical and Diagnostic Pathology

ISSN (P): 2617-7226
ISSN (E): 2617-7234
Impact Factor (RJIF): 6.3
www.patholjournal.com
2025; 8(4): 21-26
Received: 12-09-2025
Accepted: 15-10-2025

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Study of blood coagulation abnormalities in patients with liver diseases in Al-Nasiriya City, Iraq

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DOI: https://www.doi.org/10.33545/pathol.2025.v8.i4a.2103

Abstract

Background: Liver diseases pose a major public health problem both globally and locally. Patients with hepatic dysfunction often have coagulation abnormalities, which are an important component of morbidity and mortality. This study aimed to evaluate the incidence and patterns of blood coagulation abnormalities in patients with different liver diseases in the Al Nasiriya part of Iraq.

Methods: A cross-sectional study was conducted on 150 patients with documented liver diseases attending a Al Hussain teaching hospital in Nasiriya from March 2023 to February 2024. Each of the patients was evaluated using clinical assessment and laboratory investigations including prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR), platelet count and fibrinogen levels. The severity of disease was stratified using Child-Pugh classification.

Results: Out of the 150 patients (mean age 52.3±11.8 years; 65% male), most were found to have coagulation abnormalities (87.3%). Most frequent abnormality was increased PT (78.7%) closely followed by increased INR (71.3%) and thrombocytopenia (64.0%). There was a close association found between the severity of disease and the coagulation profile parameters. The most common etiology of liver disease was the hepatitis C virus (52%), followed by hepatitis B virus (28%) and alcoholic liver disease (20%).

Conclusion: Coagulation abnormalities are very frequent in patients with liver diseases. There is a close correlation between liver disease severity and coagulation abnormalities. Therefore, regular monitoring of coagulation profile parameters is important in risk stratification and management of complications. The findings of this study strongly suggest the value of extensive coagulation screening in patients with liver disease in order to institute appropriate therapeutic measures and prevention of bleeding complications.

Keywords: Coagulation abnormalities, liver disease, cirrhosis, prothrombin time, hepatic dysfunction, thrombocytopenia, coagulopathy, hemostasis

Introduction

Liver disease encompasses a spectrum of pathological processes from acute hepatitis to chronic cirrhosis with end-stage liver failure. The liver is the principal organ responsible for maintaining hemostasis through the synthesis of nearly all coagulation factors, including prothrombin, fibrinogen, and factors V, VII, IX, X, XI, and XII, with the notable exceptions of von Willebrand factor and factor VIII [1]. Thus, hepatic dysfunction invariably produces marked changes in the coagulation cascade, resulting in a complex state of dyscoagulability with both thrombotic tendencies and bleeding diatheses [2]. The incidence of coagulopathic states in patients with liver disease has been widely established in the international literature; however, there is an unfortunate dearth of epidemiologic data from Iraq. The Middle Eastern area, of which Iraq is a part, suffers a considerable problem with viral hepatitis, especially hepatitis B virus (HBV) and hepatitis C virus (HCV), which are the major causes of chronic liver disease and cirrhosis in this part of the world [3].

Disorders of hemostasis in patients with liver disease are multifactorial in origin and have both quantitative and qualitative defects. The primary mechanism is due to the decreased hepatic synthesis of procoagulant factors, including fibrinogen, factors II, V, VII, IX, X, XI, and XII. Patients with liver disease also have elevated fibrin degradation product levels, thrombocytopenia due to bone-marrow suppression and splenic sequestration, dysfunctional platelets, increased fibrinolytic activity, and endothelial dysfunctions [4]. Portal hypertension, a characteristic of advanced liver disease, results in splenic engorgement and hypersplenism, and, hence, a further decreased platelet count predisposes the severity of coagulopathy and

correlates fairly well with the degree of hepatic synthetic injury and total disease severity in prognostic scoring systems [5]. The usual laboratory measures of coagulation used in the individual tests, such as Prothrombin Time (PT), activated partial thromboplastin time (aPTT), and platelet count, are regarded as the primary tests for screening use in clinical circumstances. The PT provides a direct indication of the extrinsic path of coagulation and the common path and is therefore a highly sensitive indicator of hepatic synthetic injury. The international normalized ratio (INR), derived from the PT, allows for the standardization of the various tests and is particularly helpful in the follow-up of anticoagulant therapy. The clinical analysis of these tests in the setting of liver disease deserves scrutiny, as the normal values for these tests in their various degrees of abnormality have possibly lost their correlation to bleeding in this setting [6].

The increased INR and protracted PT have been felt to be predictive of bleeding for considerable time intervals, but more recently the emerging data tends to suggest that the cirrhotic retains a rebalanced milieu of coagulation despite the markedly abnormal results of the routine coagulation tests. The clinical implications of hepatic coagulopathy are manifest and varied. Spontaneous bleeding complications, particularly gastrointestinal bleeding from esophageal varices, portal gastropathy, or ectopic varices, are one of the most serious complications of advanced liver disease, with reported mortality rates of more than 30% in some series [7]. Paradoxically, however, patients with cirrhosis are at increased risk for thromboembolic complications, with portal vein thrombosis noted in approximately 25% of patients with cirrhosis and splenic vein thrombosis in as many as 15% [8].

This procoagulant state is thought to occur secondary to decreased anticoagulant production, endothelial dysfunction, decreased perfusion, and platelet activation. Thus, the balance between bleeding complications and thrombotic complications represents a significant challenge in the management of patients with liver disease. Al-Nasiryah, in the Dhiqar Governorate in southern Iraq, has considerable problems with health care including endemic viral hepatitis. The health care infrastructure in Al-Nasiryah includes several private clinics that provide health care to the local populace with various diagnostic and therapeutic abilities. Knowledge of the local epidemiology of the various coagulation disorders in patients with liver disease is vital for optimizing patient management and the appropriate allocation of resources in this region [9]. There have been several studies from other areas in Iraq as well as from neighboring countries, which have demonstrated a high prevalence of viral hepatitis related liver disease; however, there are limited data concerning the adequate description of coagulation perioperative abnormalities [10]. The treatment of the coagulation abnormalities in patients with liver disease requires an all-inclusive approach, including treatment of the underlying liver disease, prophylaxis against variceal hemorrhage by use of beta-adrenergic receptor antagonists and endoscopic therapy, careful use of blood products, and monitoring for thrombotic complications [11]. Recent advances in the understanding of the hepatic coagulopathy have emphasized recommendations against the prophylactic correction of routine elevated prothrombin time PT or elevated INR levels in the absence of active hemorrhage. Specific clinical situations, however, such as invasive procedures planned, may warrant temporary corrections. The determination and validation of hepatology-specific tests for hemostasis may improve risk stratification beyond the traditional tests [12].

The overall objectives of this study were to assess the prevalence of and types of coagulation disturbances present in patients with liver disease seen in a private medical clinic in the city of Al-Nasiryah, to determine correlations between coagulation tests and liver disease severity and to set baseline epidemiological data that will aid clinical decision making and be helpful in radiologically oriented therapeutic intervention. This project will contribute toward filling the gap in the local literature regarding the hepatic coagulopathies and also furnish evidence-backed data to support clinical management protocols in the private health care setting in Al-Nasiryah.

Methodology

Study Design and Setting

This investigation was a cross-sectional observational study conducted in a Al Hussain teaching hospital in Nasiriya city, Thi Qar Governorate, southern Iraq. The period of the study extended from March 2023 to February 2024, over a period of one year, in order to be able to assess seasonal variations and, at the same time, get a reasonable sample size. The private clinic is a secondary health institute where the laboratory work and imaging facilities are at their full scope. All the procedures performed in the study were approved by the ethics committee of the private clinic, and written informed consent was taken from the participating patients before entry into the study.

Study Population

The study examined 150 consecutively studied patients (over 18 years of age) having clinical liver disease and being seen in the clinic between the dates on which the study was conducted. To be included patients had to belong to the following designated groups of liver disease (diagnosis of liver disease proved by clinical findings, blood and x-ray examinations: cirrhosis, or chronic active hepatitis, or acute hepatitis, or fatty liver disease); written informed consent obtained from patients; and complete laboratory data available.

These were the exclusion criteria: Patients with primary coagulation defects (not due to liver disease), acute thromboembolic disease or on anticoagulants, having had blood transfusion or fresh frozen plasma (within past two weeks), all patients on any antiplatelet agents or anticoagulants, having malignancy with active chemotherapy, all sepsis or acute infected patients requiring hospitalization, incomplete laboratory data. Pregnant patients and those with renal failures requiring kidney dialysis were also excluded in order to eliminate confused variables interfering with coagulation parameters.

Data Collection and Clinical Assessment

Extensive demographic and clinical information was obtained including age, sex, duration of liver disease, symptoms at presentation, history of variceal bleeding, ascites and hepatic encephalopathy episodes. A set of standardized questionnaires was used to assess alcohol consumption, blood transfusion history and previous medical interventions. Physical examination findings including jaundice, ascites, splenomegaly, spider angiomas and hepatomegaly were noted. Clinical severity was evaluated using the well-accepted Child-Pugh classification

system consisting of 5 parameters: serum bilirubin, serum albumin, prothrombin time/INR, ascites and hepatic encephalopathy. The patients were categorized in three classes; (Child Pugh A score 5-6, mild), Child Pugh B score 7-9 moderate, Child Pugh C score 10-15 severe) [13].

Laboratory Investigations

Blood samples were obtained by venipuncture in the morning after overnight fasting. For the coagulation studies, the blood was drawn into sodium citrate tubes (3.2% sodium citrate, 9:1 v/v ratio) for the prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen tests. For the platelet counts and other hematological parameters, EDTA tubes were used. Complete liver function tests were made, including serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase, gamma-glutamyl transferase (GGT), total and direct bilirubin, serum albumin and total protein. Renal function tests, including serum creatinine and blood urea nitrogen (BUN), were also measured along with electrolytes. The coagulation parameters measured comprised prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR), platelet count, and fibrinogen levels.

Tests for serological evidence of hepatitis (HBsAg, anti-HBc, anti-HCV, and anti-HAV IgM) were made by using standardized immunoassays. All laboratory investigations were performed using standardized techniques in the accredited laboratory of the clinic. The coagulation studies were performed using automated coagulometers (Sysmex CA-7000 series), which were calibrated each day using control samples. Normal reference ranges: PT 11-13.5 seconds, aPTT 25-35 seconds, INR 0.8-1.1, platelet counts 150,000-400,000 cells/μL, fibrinogen 200-400 mg/dL. Liver biochemistry and other hematological parameters were analysed using automated chemistry analyzers (Beckman Coulter AU5800) with manufacturer supplied reference ranges. The quality control procedures included daily calibration, bi-monthly checking with control materials, and participation in external quality assurance programmes.

Imaging Studies

All patients underwent abdominal ultrasound scanning using a standard B mode ultrasound machine (GE Logiq E9) handled by trained radiologists. The ultrasound findings evaluated included the echotexture of the liver (normal, fatty liver infiltration, cirrhotic), the liver size (normal, enlarged or atrophic), spleen size, presence of ascites, diameter of portal vein, patency of hepatic vein and signs of portal hypertension [14]. Computed tomography (CT) scanning with intravenous contrast was performed in selected patients (N=45) with significant diagnostic uncertainty and/or suspicion of hepatocellular carcinoma in order to confirm diagnosis and define disease extent.

Diagnostic classification of liver disease

The etiology of liver disease was determined from clinical, serological, imaging, and if available, histological data. Viral hepatitis B was diagnosed from positive HBsAg and anti-HBc. Viral hepatitis C was diagnosed from positive anti-HCV antibody confirmed by detection of HCV RNA using polymerase chain reaction. Alcoholic liver disease was diagnosed, in the presence of characteristic clinical and biochemical findings (elevated GGT to alkaline phosphatase ratio etc.), from significant consumption of alcohol (≥ 40 g/day for men, ≥ 20 g/day for women for at least 5 years). Fatty liver disease was diagnosed from imaging findings of fatty infiltration without other identifiable etiology or significant alcohol consumption. Autoimmune hepatitis, hemochromatosis and other rare causes were diagnosed from serological markers and clinical findings [15].

Statistical Analysis

Data were analyzed with (Statistical Package for the Social Sciences) version 25.0. Descriptive data are expressed as mean with standard deviation for continuous normal variables, median with interquartile range for non-normal continuous variables, and as a frequency count with percentage for categorical variables. Normality was assessed by the Shapiro-Wilk test. Univariate analysis was performed to compare coagulation parameters between groups using independent samples t tests for normal variables and the Mann-Whitney U test for non-normal data. The association between categorical variables was evaluated using Chi-squared analysis. Correlation analysis was done between coagulation parameters and continuous clinical variables by means of Pearson's and/or Spearman's coefficients, as appropriate. Multivariate linear regression analysis was used to assess for significant independent variables associated with PT prolongation and INR increase. A p-value of < 0.05 was considered to be statistically significant. Receiver Operating Characteristic (ROC) curves were generated to assess the diagnostic accuracy of coagulation parameters in predicting disease severity.

Results

Patient Demographics and Clinical Characteristics

In the study, a total of 150 patients with liver disease were evaluated. The mean age of the patients was 52.3±11.8 years (23 to 78 years), were 65% male and 35% female with a male to female ratio of 1.86: 1. The mean duration of the known liver disease was 6.2±4.1 years. With respect to the etiology of the disease the most common was hepatitis C virus (HCV) infection in 78 patients (52%) followed by hepatitis B virus (HBV) in 42 patients (28%) alcoholic liver disease in 21 patients (14%) and non-alcoholic fatty liver disease (NAFLD) in 9 patients (6%). Among the HCV positive patients 62 patients (79.5%) had genotype 1 and 16 patients (20.5%) had genotype 3. The patients of HBV were further subdivided into HBeAg positive (31 patients, 73.8%) and HBeAg negative (11 patients, 26.2%). In respect to the clinical presentation, 98 patients (65.3%) had established cirrhosis and 52 patients (34.7%) had chronic hepatitis without evidence of cirrhosis.

Table 1: Demographic and Clinical Characteristics of Study Population (N=150)

Parameter	Number (n)	Percentage (%)
Gender-Male	97	64.7
Gender-Female	53	35.3
HCV Positive	78	52.0
HBV Positive	42	28.0
Alcoholic Liver Disease	21	14.0
NAFLD	9	6.0
Cirrhosis Present	98	65.3
Chronic Hepatitis Only	52	34.7

Coagulation Parameters

Abnormalities of coagulation were seen in 131 patients (87.3% of total 150 patients studied). The most frequently abnormal laboratory finding was prolonged prothrombin

time (PT) which was present in 118 patients (78.7% of the patients studied). The mean PT was 16.8±4.2 seconds (normal 11-13.5 seconds). The elevated international normalized ratio (INR) was present in 107 patients (71.3%), the mean INR being 1.8±0.6 (normal 0.8-1.1). The prolonged activated partial thromboplastin time (aPTT) was present in 89 patients (59.3%) and the aPTT was 42.1±8.4 seconds (normal 25-35 seconds). Thrombocytopenia (platelet count < 150,000 cells/mm3) was present in 96 patients (64.0%), the mean number of platelets being 128,400±56,200 cell/ mm3 (normal 150,000-400,000 cells/mm3). Hypofibrinogenemia (fibrinogen < 200 mg/dL) was the least commonly seen abnormality, being present in 38 patients (25.3%), with a mean fibrinogen level of 256±94 mg/dL (normal 200-400 mg/dL).

Table 2: Coagulation parameters in study population. PT: Prothrombin time; INR: International normalized ratio; aPTT: Activated partial thromboplastin time; SD: Standard deviation

Coagulation Parameter	Abnormal n (%)	Mean ± SD	Normal Range
PT (seconds)	118 (78.7%)	16.8±4.2	11-13.5
INR	107 (71.3%)	1.8±0.6	0.8-1.1
aPTT (seconds)	89 (59.3%)	42.1±8.4	25-35
Platelets (×10³/μL)	96 (64.0%)	128.4±56.2	150-400
Fibrinogen (mg/dL)	38 (25.3%)	256±94	200-400

Coagulation abnormalities by disease severity

Stratification according to Child-Pugh classification revealed that the severity of liver disease was related to the coagulation tests. Of 98 cirrhotic patients classified according to Child-Pugh criteria (A-18 patients, mean PT 14.2±2.1 sec; INR 1.3±0.2; B-45 patients, mean PT 16.1±3.4 sec; INR 1.7±0.4; C-35 patients, mean PT 19.4±4.8 sec; INR 2.4±0.7), the average platelet count was 156,000±32,000/μL in Child-Pugh A patients, 128,000±48,000/μL in Child-Pugh B, and 94,000±58,000/μL in Child-Pugh C patients. All of the differences were statistically significant (p<0.001). Non-cirrhotic patients with chronic hepatitis (N=52) had coagulation abnormalities, but these were less frequent than in cirrhotic patients, i.e., 62 per cent of patients had prolonged PT and 48 per cent had increased INR compared with 89 and 78 per cent, respectively, in cirrhotic patients.

Table 3: Coagulation Parameters by Child-Pugh Classification in Cirrhotic Patients. Values expressed as Mean ± SD. PT: Prothrombin time; INR: International normalized ratio

Child-Pugh	N	PT (sec)	INR	Platelets (×10³)
A (N=18)	18	14.2±2.1	1.3±0.2	156±32
B (N=45)	45	16.1±3.4	1.7±0.4	128±48
C (N=35)	35	19.4±4.8	2.4±0.7	94±58

Discussion

This cross-sectional study of 150 patients with liver disease attending a Al Hussain teaching hospital in Nasiriya is one of the first of its kind to comprehensively assess the coagulopathy seen in hepatic dysfunction within the local community. It shows that coagulopathy is common in liver disease, being found in 87.3 percent of the study population, which is commensurate with the incidence of 80-90 percent reported in the international literature [16]. The high incidence of coagulopathy which has been found in this study brings out the universal application of hepatic coagulopathy and indicates the necessity for its consideration as a clinical entity in the management of liver disease, irrespective of locality and whether the patient is in a hospital or private practice.

The most common laboratory abnormality found in this study was prolonged prothrombin time, being found in 78.7 percent of the patients studied. The prothrombin time indicates the function of the extrinsic (factor VII) and common (factors II, V, X and fibrinogen) coagulation pathways, and is therefore very sensitive to impaired function due to hepatic failure [17]. The mean prothrombin time of 16.8±4.2 seconds which we found is far above the normal upper limit and indicates that there is a significant impairment of the hepatic synthetic function. An elevated INR was found in 71.3 percent of the patients with a mean of 1.8±0.6, which is well above the limit which in other series has been shown to give an indication of possible bleeding risk (INR > 1.5) in non-liver disease patients. But it is now recognised that an elevation in the INR in cirrhosis does not afford a reliable guide to the possibility of bleeding occurring because, with the diminished levels of both procoagulation and anticoagulation factors, this dichotomy leads to a prothrombotic state which has been adjusted. Therefore, the problem of INR elevation need not concern us at the moment, because a bleeding tendency need not be anticipated in this situation [18].

Thrombocytopenia was found in 64 percent of the patients seen in this study, with a mean platelet count of 128,400 cells/μL, and is similar to that reported in overseas series.The mechanisms behind thrombocytopenia in liver disease are multifactorial. There is direct primary bone marrow suppression by alcohol in patients who have alcoholic liver disease, direct hepatitis B and C virus induced effects on megakaryopoiesis, and portal hypertension induced splenic sequestration all contribute to decreased platelet levels [19]. Portal hypertension with splenomegaly leads to sequestration of platelets in the enlarged spleen, with a correlation of splenic vein size and spleen size to platelet count in our group (r=-0.64, p<0.01). Importantly, platelet count has an important prognostic importance, with progressive thrombocytopenia correlating closely with increased severity of disease and an increased risk of variceal hemorrhage [20].

The close correlation that we documented between severity of liver disease and coagulation parameter abnormality confirms the use of coagulation tests in prognostic assessment. Child-Pugh C patients had much more profound coagulation abnormalities than Child-Pugh A patients, having PT differences of 5.2 seconds and INR differences of 1.1 unit. These differences were statistically significant (p<0.001) and clinically significant. Similar trends have been documented in studies in Europe, Asia, and the Mid-East, and tend to show that the relationship between liver synthetic functional impairment and coagulation parameters is a universal finding in the pathophysiology of cirrhosis of the liver [21]. The great correlation between coagulation parameters and severity of disease lends itself to continued application of these tests as elements of prognostic scoring systems like the Child-Pugh and Model for End-stage Liver Disease (MELD) test. There were slight differences in the etiology of liver disease in our group compared to the worldwide patterns. Infection with the hepatitis C virus was predominant in our series at 52%, which is greater than the global average of 35%, reflecting the marked endemicity of HCV in Iraq and in the Middle East generally. Infections with the hepatitis B virus were responsible for 28% of cases, in comparison with 25% globally [19].

Alcoholic liver disease accounted for 14% of our cases, which is less than is found in the developed Western countries, in which alcohol is responsible for 35-40% of cases of cirrhosis [20]. Non-alcoholic fatty liver disease (NAFLD) affected 6% of our cohort, which is significantly less than is found in the global population, and indicates that metabolic factors and obesity, though increasingly prevalent, have not yet assumed the proportions seen in European countries. The prominence of viral hepatitis in our area indicates an inadequate level of public health preventive measures, such as poor immunization levels and inadequate blood transfusion screening programs. Hypofibrinogenemia was the least common coagulation disturbance noted in our series, being found in 25.3% of patients, whereas it is found in 20-50% of patients in international series. Fibrinogen is synthesized solely in the liver, and its amount present is a measure of the synthetic capacity of the liver. The mean value of fibrinogen of patients in the study, in the proportion of 256 mg/dl, suggests that in fact, most patients were able to maintain sufficient levels of fibrinogen to support hemostasis, though in the lower part of the range of normal. 21 Interestingly enough, only 25% were found to have levels below 200 mg/dl, suggesting that synthesis of fibrinogen is relatively preserved, in fact, in comparison with the synthesis of other factors. This selective preservation of fibrinogen may relate to feedback mechanisms that promote fibrinogen production as other factors become depleted, or to an intrinsic feature of the regulation of fibrinogen gene expression in hepatic injury [25].

In our study the non-cirrhotic chronic hepatitis patients formed a clearly identifiable group, with generally less severe biochemical coagulation abnormalities. Of the 52 non-cirrhotic patients, only 62% had prolongation of the PT, compared with 89% occurrence in cirrhotic patients. This is an important clinical distinction as it implies that the presence of cirrhosis rather than mere chronic hepatitis caused the coagulation dysfunctions. This is in accord with the understanding that cirrhosis is a qualitatively different degree of liver dysfunction, characterized by architectural derangement and portal hypertension, both of which contribute to a hypercoagulable state apart from mere hepatocellular inflammation alone [26]. The clinical management implications of our study are very significant. Presently guidelines recommended against the routine prophylactic correction of coagulation parameters in asymptomatic patients with liver disease, in as much as the bleeding risk is not as severe as the usual PT and INR might suggest. Also that correction of these values carries with it the risks of transfusion-related complications and promotion of thromboembolic phenomena [27].

Our data suggests that there is a need for careful individualised assessment of, and monitoring of coagulation parameters in all persons with liver disease, more particularly so if there are signs of portal hypertension, in other words those patients where the regulation of the coagulation parameters is most compromised, as these are similar findings to those obtained in the study of the clinical effects of hypercoagulation on severity of liver disease and complication risk. Specific clinical situations would suggest more active methods of intervention, namely, patients undergoing invasive procedures, where it might be necessary to temporarily correct coagulation parameters [28]. The private clinic setting of this study reflects the patterns of healthcare delivery in the Alnasiryah area and throughout Iraq, where a significant volume of diagnostic services for liver disease patients is performed in private healthcare establishments. Our findings also provide baseline data of relevance to those private healthcare providers managing liver disease patients. The incorporation of coagulation assessment into the routine evaluation of opened liver disease, patients attending private clinics should enhance risk stratification and also facilitate appropriate referrals to specialist management. Prospective multicentre studies and more sophisticated coagulation assessment methods such as thromboelastography are needed to further enhance the evidence base and understanding of the rebalanced coagulation state in cirrhotic patients.

Conclusion

Coagulation disorders are very common in patients with liver disease who presented in private clinics in Alnasiryah City, Iraq, with a prevalence of 87.3% in the study population. Prolonged prothrombin time and elevated international normalized ratio were the most important types presented with a significant relation to the severity of disease as described by Child-Pugh classification. The etiologic factors were found to be hepatitis C virus and hepatitis B in 80% of cases in this population. Periodic evaluations of coagulation parameters is essential for an appropriate segmentation of risk group establishment, prognostication and management of patients with hepatic disease. The application of a comprehensive coagulation screening programme should be instituted in the routine clinical evaluation of all patients documented or suspected of liver disease. Our results give important local epidemiological information which will alow the implementation of evidence based clinical management protocols and give a supportive rationale for the vital need of further studies on therapy so that hepatic coagulopathy may be optimally treated in this area

Conflict of Interest
Not available

Financial Support
Not available

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How to Cite This Article
Khudhair MY. Study of blood coagulation abnormalities in patients with liver diseases in Al-Nasiriya City, Iraq. International Journal of Clinical and Diagnostic Pathology. 2025;8(4):21-26.